Commentary on "A National Longitudinal Study of the Psychological Consequences of the September 11, 2001 Terrorist Attacks: Reactions, Impairment, and Help-Seeking"

Fighting the Psychological War on Terrorism

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The article by Stein and associates (Stein et al. 2004) documents the psychological consequences of the September 2001 terrorist attacks on the World Trade Center. A significant number of the 16% surveyed adults with persistent distress reported ongoing impairment in vocational and social functioning as well as alcohol/medical/drug misuse. We are particularly concerned that so few informants either received counseling or were provided with pertinent information about posttraumatic distress from general medical providers or other sources.

Life in the United States has changed since September 11, 2001. National fears of bioterrorism have fueled the establishment of the Department of Homeland Security, military actions in Afghanistan and Iraq, and increased security at airports, schools, and landmarks deemed potential terrorist targets. But terrorism is primarily psychological warfare. The impact of terrorism extends far beyond the immediate victims of such attacks. Its primary goal is to produce widespread social disruption through creation of a credible threat affecting cognitive, emotional, and behavioral stability and function. Indeed, the major changes in public policy and government expenditures since September 11, 2001, are clear evidence that the attacks on the World Trade Center and Pentagon have continued to exert a profound psychological impact on American society as a whole.

There is no question about the urgent need for prevention and protection against attacks with chemical, biological, nuclear, or even conventional weapons. We are concerned, however, that there has been insufficient attention to the major weapons wielded by terrorists. Those weapons are fear and anxiety. They are highly toxic, infectious agents that can bring a nation or society to its knees. Contagion by fear or anxiety can be immediate. It can infect large areas of the

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population that were never in any danger from the particular weapon utilized in a specific terrorist attack. And they can have lethal consequences. During the 1991 Gulf War, 11 Israelis, who were never in physical danger from Iraqi SCUD missiles, died when air raid alerts were sounded, 7 by suffocation from faulty use of gas masks and 4 from heart attacks (Bleich et al. 1992).

The September 11th terrorist attacks were a horrible wake-up call for clinical and scientific members of the disaster mental health community. When civil leaders and policy-makers turned to us for advice, we had few recommendations to offer that were solidly supported by scientific evidence. We had no suggestions for risk communication strategies that might prevent an epidemic of fear or anxiety throughout the population as a whole. In addition, research about the best ways to detect the most vulnerable populations and about the most effective early interventions for treating adults and children was both inadequate and controversial.

Recognition of the flimsy scientific basis for practice guidelines concerning any large-scale disaster had prompted federal mental health officials (from the Departments of Defense, Veterans Affairs, Justice, and Health and Human Services) to begin planning a consensus conference almost a year before the World Trade Center and Pentagon attacks. That assembly of 80 national and international experts was convened six weeks after September 11th. Its proceedings are available on the Internet (http://www.nimh.nih.gov/research/massviolence.pdf) and in a booklet published by the National Institute of Mental Health (NIMH Report 2002). The focus was practical. A surprisingly high degree of agreement was achieved on key operating principles, timing, screening, effective early interventions, surveillance, research, and ethical issues. It is an excellent document by which to gauge the current beliefs of the world's experts on the best intervention strategies following mass casualties or national disasters.

It was also necessary to take a careful and thorough inventory of our scientific understanding about the human stress response, about normal recovery versus pathological and chronic consequences from acute traumatization, about psychological and biological models of resilience versus vulnerability, about evidence-based effective early interventions, and about important differences between children and adults. To that end the Anxiety Disorders Association of America (ADAA) convened a conference of scientific experts to review the current state of evidence concerning acute stress reactions following a major catastrophe. Three separate panels of experts were assigned to consider questions regarding epidemiology/phenomenology, psychological/biological models, and evidence-based early interventions. Specific papers from that conference along with our editorial comments have been published elsewhere (Bryant 2003; Cohen 2003; Ehlers and Clark 2003; Friedman, Foa, and Charney 2003; March 2003; McNally 2003; Morgan et al. 2003; Norris, Murphy, Baker, and Perilla 2003; Pine 2003). They serve as the detailed literature reviews on which we base our present remarks. The purpose of this commentary is to synthesize the published manuscripts, formal reports, and group discussions that occurred at the ADAA conference within the wider context of the published literature on acute traumatic stress. Our approach is to present a status report on current scientific theory and evidence on acute stress responses following catastrophic events. We emphasize normal as well as pathological reactions. In addition to a comprehensive but succinct overview, we will propose a scientific agenda for advancing the psychological war against terrorism.

EPIDEMIOLOGY/PHENOMENOLOGY: IDENTIFYING SURVIVORS AT RISK

Epidemiology

A synthesis of 160 studies on disaster victims indicates that among high impact disasters, a third or more survivors experience clinically significant distress. Delayed onset is rare and survivors at greatest risk for

long-term impairment are most likely to express such symptoms during the acute aftermath of the trauma (Norris et al. 2002a, b). Individual-level risk-factors for poor mental health outcomes include: a) severity of exposure; b) personal characteristics (e.g., female gender, middle-aged adult, ethnic minority status, low socioeconomic status, previous psychiatric history, little previous disaster exposure); c) family context (child caring responsibilities for females, parental distress for children, significant distress by any family member); and d) resource loss (13).

Diagnostic Issues

The Diagnostic and Statistical Manual fourth edition (DSM-IV) (American Psychiatric Association 2000) attempted to identify acutely traumatized individuals at greatest risk to develop posttraumatic stress disorder (PTSD) by including a new diagnosis, Acute Stress Disorder (ASD). The core symptoms that differentiate ASD from PTSD are dissociative, despite very little evidence justifying their prominence as diagnostic criteria. Although it has been shown that a significant number of individuals (approximately 75%) who develop ASD within the first month after a trauma will develop PTSD, it has also been shown that the majority of individuals who develop PTSD have never met criteria for ASD (Bryant 2003). Thus, ASD fails to identify a significant portion of individuals requiring treatment.

Similarly, no biological predictors of PTSD development have been definitively identified. Preliminary reports have suggested that elevated heart rate and inadequate cortisol levels have predictive ability (Bryant, Harvey, Guthrie, and Moulds 2000; Shalev et al. 1998) but contradictory findings exist (Blanchard, Hickling, Golavski, and Veazey 2002).

Posttraumatic panic attacks may be a useful predictor of PTSD occurring in 53 to 90% of traumatized individuals (Bryant and Panasetis 2001; Nixon and Bryant, 2003; Resnick, Falsetti, Kilpatrick, and Foy 1994). Indeed, the recent study by the New York Academy of Medicine found that after the attacks on the World Trade Center, the occurrence of a panic attack was an excellent predictor of the subsequent development of PTSD (Galea et al. 2002).

Research with Children

In the few studies that have looked specifically at ASD symptoms in children and adolescents (Daviss et al. 2000; Fein et al. 2002; Winston et al. 2002), there is little evidence that the full syndrome is a useful construct for this age group or that it is a good predictor of subsequent PTSD (March 2003). In the only prospective study, ASD was a poor predictor since only 12% developed PTSD at follow-up (Daviss et al. 2000). In addition, published research with children and adolescents has not paid sufficient attention to specific stressor characteristics, developmental factors, or level of parental stress (March 2003). Thus, to date, there have been too few longitudinal studies with children from which to draw any firm conclusions.

The Next Steps: Epidemiology/Phenomenology

In examining the literature on the epidemiology and phenomenology of PTSD, it is clear that there are still a number of key gaps in our scientific understanding.

- More information is needed on the epidemiology of posttraumatic reactions in the acute phase.
- We also need to learn how to distinguish resilient individuals for whom normal recovery can be expected from vulnerable individuals for whom more aggressive early interventions are needed.
- Age, gender, and cultural differences need to be identified as well as differences that may exist based on the characteristics of the trauma. This is especially true in the case of children and adolescents.
- Standardized measures and methods for studying trauma populations need to be developed.

Separate initiatives will be needed for research on children because of the unique developmental and methodological challenges they present. In short, we agree with the recommendations of experts who attended the ADAA conference that we must develop conceptual models to characterize the range of acute phase reactions and to predict chronicity. This can be accomplished by utilizing epidemiological research on the general population and by conducting more focused investigations on specific vulnerable and/or resilient groups.

PSYCHOLOGICAL AND BIOLOGICAL MECHANISMS

Psychological Mechanisms

Studies of children and adults exposed to a variety of adverse conditions including war, family violence, and natural disasters have revealed a set of individual characteristics that have been associated with successful adaptation to extreme stress. These include good intellectual functioning, effective self-regulation of emotions and attachment behaviors, positive self concept, optimism, altruism, a capacity to convert traumatic helplessness into learned hopefulness, and an active coping style in confronting a stressor (Bell 2001; Grinker and Speigel 1945; Masten and Coatsworth 1998; Rachman, 1992; Richardson 2002; Ruff and Korchin 1964) with greater ability to process traumatic information adaptively.

It has been suggested that higher intelligence protects against PTSD because it is associated with greater problem-solving ability and greater ability to process traumatic information adaptively, both of which may be keys to recovery (Amir, Stafford, Freshman, and Foa 1998; Cohen 2003; Silva et al. 2000).

Differences in posttraumatic perceptions of self and world may be another cognitive mechanism that may distinguish resilient from vulnerable survivors. Two basic dysfunctional cognitions have been proposed to mediate the development and maintenance of PTSD after a traumatic event: the world is completely dangerous and one's self is totally incompetent (Foa & Cahill 2001). In addition, perceived threat (Kilpatrick et al. 1982), negative expectations regarding the immediate and long-term impact of the traumatic event (Bryant 2003; McNally 2003), and a sense of personal incompetence and loss of sanity all predict PTSD (Ehlers and Clark 2000; McNally 2003), as do posttraumatic feelings of shame, guilt, and self-blame (Andrews, Brewin, Rose, and Kirk 2000; McNally 2003).

If negative cognitions are associated with PTSD, it would be expected that positive cognitions would be associated with resilience. Indeed, PTSD rates were surprisingly low among military prisoners of war (Nice et al. 1996; Sledge, Boydstun, and Rabe 1980) and Turkish political detainees (Basoglu et al. 1997) who, despite having been tortured during captivity, felt that the experience had contributed to their personal growth. In short, the presence of negative versus positive posttraumatic cognitions may help to distinguish vulnerable from resilient survivors.

Finally, there is evidence that accurate encoding, processing, and retrieval of traumatic memories in the acute posttraumatic phase may protect against subsequent PTSD (Harvey, Bryant, and Dung 1998; Moulds and Bryant 2002). This suggests that it is necessary to accurately retrieve corrective information in order to construct well-articulated trauma narratives and to combat negative cognitions if recovery is to be achieved (Bryant 2003).

Biological Mechanisms

Biological models emphasize the key role of the amygdala in coordinating the response to threat via activation of corticotropin releasing factor (CRF), which mobilizes both the locus ceruleus/norepinephrine (LC/NE) and hypothalamic-pituitary-adrenocortical (HPA) systems. The human stress response is designed for both rapid reactivity and rapid termination with the amygdala setting things in mo-

tion and the medial prefrontal cortex (PFC) exercising the primary restraint function (Pine 2003). Functional brain imaging with PTSD subjects provoked by trauma-related stimuli indicates greater activation in the amygdala region with significantly reduced function in the PFC region (Bremner et al. 1999, 2000; Rauch et al. 1997). In addition, functional brain imagery suggests that PTSD is associated with noradrenergic dysregulation (Bremner et al. 1997a).

Most (Bremner et al. 1995, 2003; Villarreal et al. 2002), but not all (Bonne et al. 2001; DeBellis et al. 2001) structural imaging studies in patients with chronic PTSD have revealed reduced hippocampal volume. It is controversial at this time whether decreased hippocampal volume may represent a preexisting risk factor for PTSD (Gilbertson et al. 2002) or whether psychological stress itself reduces hippocampal neurogenesis (Gould and Cross 2002), and causes neuronal atrophy and death (Sapolsky 2000).

Human studies either with healthy subjects exposed to stress or with chronic PTSD subjects have detected abnormalities in both LC/NE and HPA systems that might have been predicted by extrapolation from animal research. These include elevated CRF in animals under stress (Chappel et al. 1990; Coplan et al. 1996) and in subjects with chronic PTSD (Baker et al. 1999; Bremner et al. 1997b). Excessive noradrenergic activity (McNally 2003; Southwick et al. 1997) and dysregulation of the HPA system has also been reported in PTSD subjects (Yehuda 2002). Other important neurotransmitter systems studied less thoroughly in PTSD subjects include opioids, serotonin, dopamine, gamma-aminobutyric acid (GABA) and excitatory amino acids (Morgan et al. 2003).

Finally, biological research with humans has pointed to the key role of N-methyl-D-aspartate (NMDA), adrenergic and serotonergic receptor mechanisms in dissociation, a symptom often exhibited by traumatized individuals (Krystal et al. 1998, 1999; Morgan et al. 2003; Southwick et al. 1997).

Animal research suggests that traumatic exposure during crucial periods in childhood may produce long-term semi-permanent alterations in brain development that affect stress response systems (Pine 2003). Changes in maternal care may also alter the development of behavioral, hormonal, and neural systems related to stress regulation (Meany 2001; Pine 2003). Finally, maternal exposure to stress may produce fearfulness, elevated CRF, and altered adrenergic mechanisms in their infant offspring (Coplan et al. 1996).

Consistent with these animal studies, structural abnormalities have been detected in the brains of maltreated children (DeBellis et al. 2002). Such abnormalities in neural development might be responsible for demonstrated impairment in traumatized children's ability to sustain attention and concentrate (Beers & DeBellis 2002; Dalgleish et al. 2001; Moradi et al. 1999) or to exhibit normal declarative memory function (Monk, Pine, and Charney 2002).

An important new area of research involves investigations designed to identify the acute neurochemical response and psychobiological neural mechanisms associated with resilience to extreme stress. Neurotransmitters, neuropeptides, and hormones such as neuropeptide Y, galanin, and dehydroepiandosterone (DHEA) may promote resilience (Charney, 2004; Morgan et al. 2001; Rasmusson et al. 2000). The neural mechanism of reward and motivation (hedonia, optimism, learned helpfulness), fear modulation (effective behaviors despite fear), and adaptive social behavior (altruism) may be relevant to the character traits associated with resilience and vulnerability to extreme stress.

The Next Steps: Psychological and Biological Mechanisms

Future research on psychological and biological mechanisms will need to operationalize and focus on adaptive versus pathological responses. Successful adaptation needs to be understood within the shifting contexts of physical survival, cognitive perceptions of the experience after survival, and readaptation to the changed environment. In

other words, adaptive responses may have to change as the context for survival and recovery shifts with the progression of events. A broad spectrum of outcomes will need to be monitored in order to assess successful adaptation, such as: functional capacity, coping with emotional distress, mental and physical health, interpersonal function, accurate perception of self and environment, ability to modulate arousal, and flexibility in response to shifting demands. Biological mechanisms, especially those mediated by the HPA, adrenergic, serotonergic, opioid, glutametergic, and gabergic systems need to be monitored systematically with a specific focus on CRF, NPY, LC/NE and glucocorticoid activity. It will also be important to understand psychological processes in terms of biological responses and biological processes in terms of psychological responses.

Three specific research strategies were recommended at the ADAA conference which we strongly endorse.

1. Conduct longitudinal studies on high risk populations (e.g., inner-city children at risk for exposure to violence) that simultaneously monitor variables related to psychological and biological mechanisms, including predictors of resilience;
2. Promote laboratory research assessing the relationship between observable symptoms and psychological and biological mechanisms; and
3. Design intervention studies to monitor psychological and biological mechanisms along with standard clinical outcomes.

EVIDENCE-BASED EARLY INTERVENTION

Psychosocial Interventions

Randomized clinical trials (RCTs) have been conducted on psychological debriefing and cognitive-behavioral therapy (CBT). A total of eleven RCTs on psychological debriefing that meet the rigorous standards of the Cochrane review of RCTs (Rose, Bisson, and Wessely 2002) have been conducted thus far. In all cases, the intervention tested consisted of a single session of individual debriefing, administered within the first month after the traumatic event. In no case did debriefing prevent the later development of PTSD (Risson et al. 2000; Rose et al. 2002) and in some cases, it appeared to delay recovery since comparison subjects who did not received debriefing were less symptomatic at follow-up (Bisson, Jenkins, Alexander, and Bannister 1997; Mayou, Ehlers, and Hobbs 2000). It is important to note that there is much more research that must be done, especially RCTs of group debriefings with groups of professional (military, firefighters, emergency medical) personnel in whom group cohesion and mutual support has already been established prior to the traumatic event.

There are theoretical reasons that might explain the consistent failure (and possibly counterproductive effect) of debriefing on recovery. Many experts (Bisson, McFarlane, and Rose 2000; Ehlers and Clark 2003; Rauch, Hembree, and Foa 2001) challenge the belief among debriefing's proponents that the intervention should be provided during the immediate aftermath of an acute trauma. They argue that early exposure to traumatic material (as part of the prescribed course of a standard debriefing) may actually interfere with a natural recovery process that allows the traumatic memory to be consolidated and then to fade from conscious awareness. Results from Ehlers and Steil (1995) suggest that contrary to what might have been expected, avoidance may be an important adaptive strategy in the very early stages of normal recovery from traumatic events that should not be disrupted by early interventions such as debriefing.

A second possible reason for the ineffectiveness of debriefing may be that such an early focus on acute posttraumatic symptoms may foster negative cognitions about oneself (e.g., "most people feel better by now so there

must be something wrong with me") (Ehlers and Clark 2003). As noted earlier, negative cognitions predict the later development of PTSD (Bryant 2003; McNally, 2003).

A third possibility proposed by experts (Ehlers and Clark 2003; Foa and Cahill 2001) to explain negative results with debriefing is that the kind of exposure that occurs in debriefing or self-help treatments may actively interfere with habituation and cognitive changes that are essential for normal recovery. This is in marked contrast with imaginal exposure that occurs as part of a CBT treatment in which therapists can control the content of the exposure to make sure that it is implemented consistently and systematically.

A fourth possibility is based on biological findings that excessive adrenergic activity predicts PTSD (Bryant et al. 2000; Shalev et al. 1998). Since noradrenergic arousal also facilitates the encoding of traumatic memories, it may be that premature activation of such mechanisms within the debriefing context fosters the encoding of intrusive memories that increase the risk for PTSD (Morgan et al. 2003).

We have gone into much detail to consider possible reasons for the consistently poor performance of psychological debriefing because this intervention is a very popular approach that is utilized extensively. For that reason, it is important to understand that there are a number of persuasive theoretical reasons to explain consistent results from RCTs showing that debriefing does not promote posttraumatic recovery and may even hinder it.

In contrast to negative findings with debriefing, at least four RCTs with CBT have had very promising results. It should be noted, in this regard, that these brief CBT interventions are usually not initiated until at least 14 days after acute traumatization, much later than the standard 72-hour posttraumatic window in which debriefing is generally offered. Brief CBT protocols of four or five sessions have been shown to ameliorate ASD or acute posttraumatic distress and to effectively reduce the subsequent development of PTSD. Brief CBT appears to have been more effective than supportive counseling, repeated assessment, or a naturalistic control group (Bryant et al. 1998; 1999; 2003; Foa et al., 1995).

Pharmacological Interventions

Given abundant evidence that excessive noradrenergic activity is associated with PTSD (Morgan et al. 2003; Southwick et al. 1997), one might expect that acute suppression of catecholamines would ameliorate acute posttraumatic distress and prevent PTSD. In the only RCT testing this hypothesis, Pitman et al. (2002) reported promising results with the adrenergic beta blocking agent, propranolol, administered to accident victims within six hours of the event. Although pharmacotherapy did not prevent the later development of PTSD, it did reduce physiological reactivity among subjects who had received treatment. Two other reports also suggest that propranolol may be an effective treatment for acutely traumatized individuals (Guillaume et al. in press; Taylor and Cahill 2002). Since enhanced noradrenergic activity may increase the likelihood of developing intrusive, emotionally arousing memories and since adrenergic blocking agents such as propranolol may suppress this process (Morgan et al. 2003), acute utilization of noradrenergic antagonists appears to be a promising area for future investigation. In addition, there is a spotty but consistent literature suggesting clinically significant results with a variety of antiadrenergic agents (e.g., propranolol, clonidine and prazosin) which have been shown to reduce PTSD reexperiencing, hyperarousal and dissociative symptoms (Friedman 2003).

The only other RCT on early intervention with pharmacotherapy involved recently traumatized children on a burn unit with ASD in which the tricyclic antidepressant imipramine produced greater reduction in ASD symptoms than the sedative/hypnotic, chloral hydrate (Robert et al. 1999). Finally, a naturalistic study with pediatric burn victims suggests that acute morphine administration during hospitalization prevented the later development of PTSD symptoms (Saxe et al.

2001). Since opiates inhibit neuronal activity in the amygdala and antagonize the actions of CRF and adrenergic neurotransmitters, this study is interesting, theoretically.

Based on aforementioned findings suggesting that dissociation can be provoked by adrenergic activators (e.g., yohimbine) serotonergic agonists (e.g., mCPP) and NMDA antagonists (such as ketamine) the field is ready for clinical trials of a variety of medications that oppose these actions such as antiadrenergic agents (especially clonidine and guanfacine), serotonin (5HT-2) antagonists and glutamatergic agents such as the anticonvulsant, lamotrigine (Friedman 2003; Morgan et al. 2003).

Treatment for Acutely Traumatized Children

To date, there are no empirical studies of psychosocial interventions for children and adolescents implemented within the first month after a traumatic event. Information on the effectiveness of treatment is largely based on three RCTs of sexually abused children, most of whom met criteria of acute PTSD. All three studies compared CBT to other treatments, nondirective supportive therapy (Cohen and Mannarino 1996, 1998), or treatment as usual (Deblinger, Lippman, and Steer 1996), and found that it was superior. Ten to 18 sessions of CBT has also been shown to be effective in treating more chronic PTSD (Chemtob, Nakashima, Hamada, and Carlson 2002; Goenjian et al. 1997; March et al. 1998). Thus, there is no information about the effectiveness of earlier and briefer interventions or what dosage may be required to be most effective.

As with adults, there are some reasons to be concerned about premature interventions with children and adolescents. First, there is no empirical evidence to support early intervention. Second, Cohen (2003) suggests that there may be developmental reasons to believe that early intervention may be harmful to children at certain ages since it might disrupt the maturation of normal coping and adaptation mechanisms. Finally, it is important to recognize that since young children base their perceptions of events on their parents' perceptions and behavior (i.e., social referencing), children may believe that they are still in danger and become more symptomatic if their parents continue to exhibit high levels of posttraumatic distress.

The Next Steps: Evidence-Based Early Interventions

Data from both adults and children suggest that attending to basic needs (e.g., safety, food, shelter, acute medical problems, etc.), psychological first aid, clinical assessment, screening, and surveillance may be the best intervention in the early phase of recovery. Treatment at this time should perhaps be more indirect, such as focusing on sleep problems, providing educational information about normal reactions to traumatic stress, encouraging survivors to seek support from significant others, and helping parents cope with their anxiety so as not to frighten their children. Focused CBT interventions may be best initiated at least several weeks and possibly several months after the trauma for those individuals still experiencing significant symptoms.

The ADAA conference experts agreed that more information is needed about interventions of all types: individual, group, and community.

- *Individual interventions* need to be well-controlled prospective trials that include a range of traumas and outcomes. CBT interventions implemented in the first month need to be compared to other treatments (e.g., psychological debriefing, pharmacotherapies, treatment as usual), repeated assessments, and the natural recovery process to determine if they are beneficial or may actually impede recovery. Such research should systematically evaluate the relative importance of treatment setting, timing of intervention, treatment dosage, population characteristics, cultural factors, and developmental level.
- Research on *group interventions* should prioritize investigations of group debrief-

ings among professional personnel who have functioned previously as a unit (e.g., military, police, firefighters, emergency medical personnel) since this very important issue has not been studied adequately. As with individual interventions, research on groups should systematically investigate timing, setting, duration/dosage, intensity of trauma exposure, and cultural factors.

- With regard to *community-level interventions*, we suggest that pretraumatic preparation and posttraumatic risk communication may be equivalent societal-level psychological approaches for combating terrorist-induced epidemics of fear. Preparation would include ongoing training and education about normal reactions to catastrophic stress and about adaptive ways of coping with terrible events. Training and education on traumatic stress could be offered routinely to students through their schools, parishioners through their churches, adults through employee assistance programs, or through many other venues. Posttraumatic risk communication would prevent rapid and widespread contagion by fear and anxiety through the provision of accurate information in a way that would resolve uncertainty and ameliorate distress.

In our opinion, the key to effective societal pre- and posttraumatic interventions is judicious and effective utilization of the mass media and Web-based technologies. We have already learned from two post-9/11 studies about the dose response relationship between exposure to repeated televised images of the World Trade Center in flames and posttraumatic distress (Galea et al. 2002; Schlenger et al. 2002). Therefore, we believe that if the media are powerful enough to intensify fear and distress throughout the population at large, there must be a rational way to harness such power to counter the psychological impact of terrorist attacks. This is a major societal priority that demands our immediate and utmost attention.

FINAL REMARKS

We agree completely with Stein and associates (2004) that a proactive public mental health strategy is needed to "provide a coordinated community-wide response for individuals needing information and counseling following terrorist events" (p. 106).

The psychological war on terrorism will require a coordinated effort by government, academic, and private institutions. Adequate resources will be needed to fund such an initiative. One specific approach might be the establishment of center grants to facilitate collaborations between the research- and disaster-response communities. Another might be pre-disaster funding that yokes research expertise with organizations that routinely respond to catastrophes (e.g., firefighters, police, military personnel, and emergency medical technicians.) A third might be priority funding for any of the research initiatives recommended above.

As stated at the outset, we believe that to wage the psychological war on terrorism we must confront the threat of terrorism from a public health perspective. We must consider psychological vaccines that foster resilience. We must figure out how to utilize the mass media to provide accurate information and risk communication in a way that will prevent widespread and counterproductive contagion by fear and anxiety but will also facilitate timely psychological intervention for people in need. And we must develop and provide effective antidotes for those who have been incapacitated or psychologically immobilized after terrorist attacks or other mass casualties.

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